



The Mechanism of Solid Phase Epitaxy

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THE MECHANISM OF SOLID PHASE EPITAXY

MICHAEL J. AZIZ
Division of Applied Sciences
Harvard University
Cambridge, MA 02138
U.S.A.

ABSTRACT. The enhancement of the solid phase epitaxial growth (SPEG) rate in Si and Ge by hydrostatic pressure, and the reduction in the rate by uniaxial compression, place severe constraints on the kinds of point defects that can be responsible for thermal SPEG. These measurements are interpreted in terms of an activation strain tensor, the nonhydrostatic analogue of the activation volume, which results from an extension of transition state theory to nonhydrostatic stress states. These results and those of other experiments allow us to rule out all mechanisms in which the rate-limiting step is thermal generation of point defects in the bulk of either phase, and the migration of these defects to the crystal-amorphous interface. All experimental results are semi-quantitatively consistent with the Spaepen-Turnbull interfacial dangling bond mechanism. The structural aspects of the Williams-Elliman interfacial kink site model are shown to be a special case of the dangling bond mechanism. The electronic aspect of the Williams-Elliman model has been generalized to take into account more recent experiments on the doping-dependence of the SPEG rate. It is compared to the fractional ionization model of Walser and Jeon. They both account for the enhancements due to low, but not high, dopant concentrations. The relevance to models for the effects of ion irradiation on SPEG is also discussed.

Introduction

Annealing of ion-implantation-amorphized surface layers of Si and Ge¹⁻⁶ results in the crystallization of the amorphous phase by solid phase epitaxial growth (SPEG), which occurs by the motion of a sharp crystal/amorphous (c/a) interface toward the free surface. The growth rates are well described by an Arrhenius dependence on temperature, with activation energies of 2.70 and 2.0 eV for Si and Ge, respectively. The effects of dopants³ and ion irradiation⁷⁻¹¹ on the growth process in Si also have been established. In spite of numerous experiments, no agreement yet exists on the atomistic mechanism of the process. Models^{2,6,12-17} invoke different types of defects whose creation or transport at or to the c/a interface are proposed as the rate-limiting step in SPEG. In Table 1 we list several models proposed to explain SPEG in Si.

Variations in pressure, like variations in temperature, are commonly viewed not to affect the nature of the atomistic processes involved, but only their rates. Thus the pressure dependence of a kinetic process, which bears directly on the atomistic mechanism, provides a unique additional parameter for its determination. Measured values of the kinetic prefactor and both the activation energy ΔE^* and the activation volume ΔV^* must be accounted for by any successful kinetic model.

TABLE 1. Candidate point defects governing crystal growth and conclusions regarding tenability of mechanisms.

Defect Governing SPEG	Authors	Conclusions
Defects residing at c/a interface		
Dangling bonds Kink sites	Spaepen and Turnbull ^a Williams and Elliman ^b	Plausible special case of dangling bond mechanism
Defects residing in crystal		
Vacancies	Csepregi <i>et al.</i> ^c , Suni <i>et al.</i> ^d	Highly Implausible (Si); Impossible (Ge)
Interstitials		Highly Implausible
Defects residing in amorphous		
Dangling bonds	Mosley <i>et al.</i> ^e	Impossible
Floating bonds	Pantelides ^f	Impossible
any other point defect in amorphous phase	Narayan ^g , Licoppe and Nissim ^h	Impossible
^a See ref. 12.	^c See ref. 2.	^e See ref. 15.
^b See ref. 13.	^d See ref. 6.	^f See ref. 16.
		^g See ref. 14.
		^h See ref. 17.

For a process controlled by the transport of a point defect present in equilibrium concentrations, ΔV^* is in many cases given by the sum of the formation volume of the defect and the volume of motion of the defect.

This manuscript summarizes two papers currently in press. Aziz, Sabin, and Lu measured the effect of uniaxial stress on the SPEG rate and extended transition state theory to nonhydrostatic stress states in order to interpret the results¹⁸. Lu, Nygren, and Aziz measured (in various combinations) the effects of hydrostatic pressure, of doping and compensation, and of structural relaxation on the SPEG rate¹⁹. Furthermore, they quantitatively examined a number of proposed mechanisms in light of the combined experimental results, showed that all bulk point defect mechanisms were inconsistent with experiment, performed a kinetic analysis of the Spaepen-Turnbull interfacial dangling bond mechanism and showed it to be in semi-quantitative agreement with all known results. They also examined the Williams-Elliman¹³ and the Walser-Jeon²⁰ models for doping dependence, and various aspects of models for the effects of ion irradiation on SPEG.

Experiment and Results

The effects of hydrostatic pressure on the solid phase epitaxial growth (SPEG) rate, v , of intrinsic Ge (100) and undoped and doped Si (100) into their respective self-implanted amorphous phases have been measured by Lu *et al.*¹⁹ Samples were annealed in a high-temperature,

high-pressure diamond anvil cell. Cryogenically-loaded fluid argon, used as the pressure transmission medium, ensured a clean and hydrostatic environment. v was determined by *in situ* time-resolved visible (for Si) or infrared (for Ge) interferometry. v increased exponentially with pressure according to

$$v = v_0 \exp\left(-\frac{\Delta E^*}{kT}\right) \exp\left(-\frac{P\Delta V^*}{kT}\right), \quad (1)$$

where v_0 is a constant, ΔE^* is the familiar activation energy, and ΔV^* is the activation volume. SPEG is characterized by a negative activation volume of -0.46Ω in Ge, where Ω is the atomic volume, and -0.28Ω in Si, as shown in Fig. 1. The activation volume in Si is independent of both dopant concentration and dopant type; no similar measurements were made in Ge.

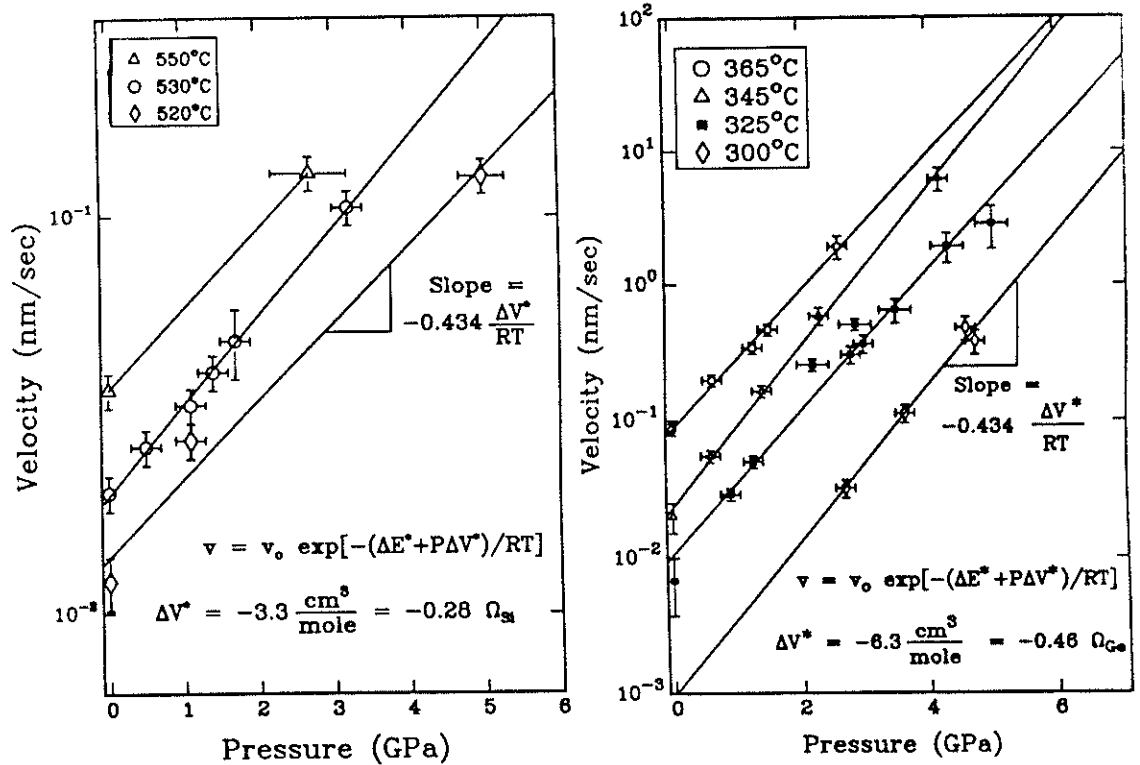


FIG. 1. Hydrostatic pressure enhancement of Solid Phase Epitaxial Growth rate in pure Si (left) and Ge (right).

Aziz *et al.*¹⁸ measured the dependence of v on uniaxial stress applied along the [100] direction in a (001) wafer. The measurement was accomplished by elastically bending Si wafers over fused quartz rods at a temperature too low for plastic deformation to occur but high enough for SPEG to proceed at measurable rates. The difference between the growth rates on the compressive and tensile sides of the wafer was measured, as was the behavior of the growth rate as the stress varied along the length of the wafer. The tensile side grew faster and the compressive side grew slower than unstressed material at the same temperature, as shown in Fig. 2. The curve fit to the data is derived from an extension of transition-state theory to nonhydrostatic stress states.

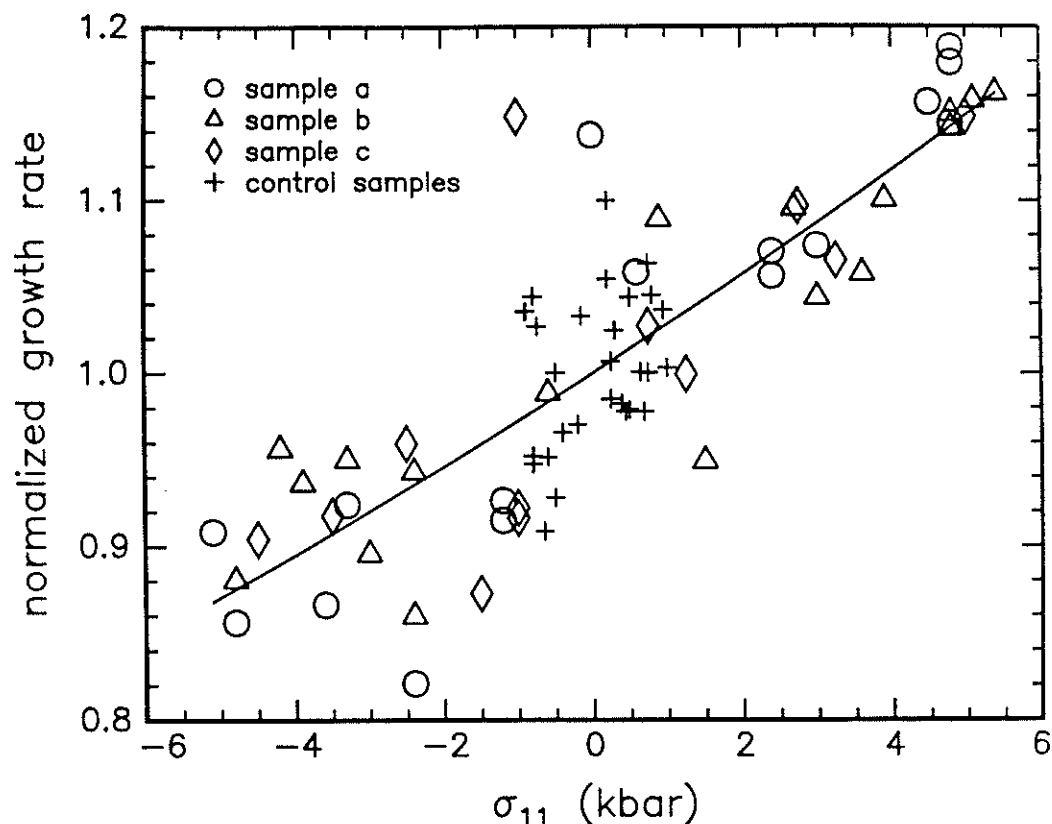


FIG. 2. Si growth rate enhanced by uniaxial tension along $[100]$ direction of (001) wafer. Positive values of the stress are tensile.

Structural relaxation affects atomic diffusion in amorphous systems²¹⁻²³. Typically, the diffusion coefficient in an amorphous phase is higher in the unrelaxed than in the relaxed state. In a study of interdiffusion in a-Si/a-Ge multilayers Park and Spaepen²² found that the diffusivity in the unrelaxed state was higher than in the relaxed state by a factor of $\sim 5-10$. Correspondingly, Lu *et al.*¹⁹ searched for an effect of amorphous relaxation on the SPEG rate in both Si and Ge from an examination of its time dependence. Comparisons were made between the SPEG rates of virgin samples and of samples that had been preannealed at lower temperatures long enough to effect nearly complete structural relaxation according to the calorimetry results of Donovan *et al.*²⁴. Care was taken that the heat-up times in the SPEG runs were much shorter than the calorimetrically-observed relaxation time at all temperatures up to the temperature of SPEG. No difference was observed between the SPEG rates of relaxed and unrelaxed samples. They concluded that structural relaxation of the amorphous phases has no significant effect on v .

Discussion

Although the SPEG process occurs by motion of a sharp c/a interface, models conflict on whether the process is controlled by interface reaction kinetics^{12,13} or by bulk diffusion of defects to the interface^{2,6,14-17}. Lu *et al.* addressed both possibilities; their analysis is summarized below.

Implications for Bulk Point Defect Mechanisms. For any bulk point-defect mechanism of thermal SPEG in which defect impingement on the interface acts in series with atomic reaction at the interface, the growth rate can be expressed by

$$v \propto \frac{k_d \cdot k_i}{k_d + k_i}, \quad (2)$$

where k_d is the rate of the bulk generation and transport of defects to the interface and k_i is the rate of reaction at the interface. Equation (2) results from DC circuit analysis, where the k 's are equivalent to inverse resistances, and the assumption of steady state is made. In kinetic processes in materials, both k_d and k_i generally have Arrhenius form. Their product is expected to have Arrhenius form if one of these rate constants is negligibly small with respect to the other (the unlikely alternative is that they have identical activation energies - and volumes). In SPEG of Si, the single activation energy over 10 decades in velocity³ offers strong evidence for a single significantly rate-limiting step. If the limiting step is the interface reaction then it is not really a bulk defect mechanism. In this case, how defects are transported to the interface becomes almost as peripheral a question as how thermal energy is transported to the atoms in the reaction. If, however, the rate-limiting step is bulk defect generation and transport to the interface, then the SPEG rate and bulk diffusivity can be directly compared. For both processes, the rate would be given by a product of the concentration of point defects, their mobility, and a geometrical factor. Hence,

$$\frac{v}{D_{\text{bulk}}} = \text{constant} \quad (3)$$

Implications for Vacancies in Ge. Recently, Werner *et al.*²⁵ produced direct evidence for the vacancy mechanism of diffusion when they found a positive activation volume in their high-pressure study of Ge self-diffusion. The clear difference between their result and the negative activation volume obtained by Lu *et al.* in Ge shows that Eq. (3) is violated, hence the defects that limit self-diffusion in the crystal and those limiting the SPEG rate are not the same.

Implications for Vacancies in Si. Despite extensive research on diffusion in Si, whether the diffusion process is mediated by interstitials or vacancies remains open. No definitive measurement exists of the activation volume for self diffusion. A recent total energy calculation by Antonelli and Bernholc²⁶ of pressure effects on Si self-diffusion yielded a vacancy formation volume of $0.75\Omega_{\text{Si}}$; this will be used to examine the predictions of a vacancy mechanism for SPEG.

If SPEG in Si were controlled by the diffusion of vacancies in the crystal, the observed pressure-enhanced SPEG would necessarily be due to pressure reducing the barrier to vacancy motion enough to overwhelm the pressure-reduced vacancy concentration. In this case, an upper limit can be placed on the formation volume of the vacancy because the barrier to motion cannot be reduced past zero without something dramatic occurring, e.g., the slope of the $\ln v$ vs. pressure isotherm might change sign. The result is a very small volume of vacancy formation, $\Delta V_f^0 \leq +0.44\Omega_{\text{Si}}$. This is much smaller than all the theoretical values of which we are aware, including the above value of $0.75\Omega_{\text{Si}}$. We therefore consider it highly implausible that in Si the formation and transport of crystal vacancies to the c/a interface is rate-limiting step for the SPEG process.

Implications for Minority Diffusers. Further considerations exist for any mechanism that involves point defects from the bulk of either phase and in which the defects contribute even a small

amount to self diffusion. By comparing v to the measured self-diffusivity, one can determine whether there are enough defects - or whether they are mobile enough - to cause the measured SPEG rate. If each such defect converts N_f atoms from the amorphous to the crystalline phase on arrival at the interface, Lu *et al.* determined an upper limit on v , which may be inverted to place a lower limit on N_f :

$$N_f \geq \frac{s^2 \lambda}{\sqrt{3}} \frac{v}{D^{\text{self}}}, \quad (4)$$

where s is the ratio of the average distance that an atom jumps during diffusion to the interatomic spacing.

Table 2 lists estimates of lower limits for N_f for the various types of defects proposed for Si and Ge SPEG. These results are based on only two major assumptions: that bulk diffusion of the defect controls the SPEG rate and that defect diffusion contributes to bulk self diffusion.

Lu *et al.* argue that values of N_f much above 10^4 seem highly implausible. They conclude it to be highly implausible that in either c-Si or c-Ge, vacancies or self-interstitials are the defects whose diffusion to the interface controls the SPEG rate. This follows simply because self diffusivities in c-Si and c-Ge are far too low to account for the observed SPEG rates.

TABLE 2. Lower limits on number of atoms crystallized per defect impingement on interface, from equation (4).

defect type	Si, 803 K	Ge, 600 K
defect in bulk crystal	$N_f > 3 \times 10^8$	$N_f > 10^8$
vacancy- and interstitial-like defects in bulk amorphous phase	$N_f > 7$	$N_f > 3 \times 10^4$
dangling and floating bonds in bulk amorphous phase	$N_f > 2$	$N_f > 1 \times 10^4$

Implications of the Lack of Relaxation in SPEG Rate. The absence of an effect of structural relaxation on v distinguishes crystal growth from diffusion in the bulk of the amorphous phase, where large reductions in diffusivity by a factor of 5 to 10 were reported²². Since this result violates equation (3), we take this as evidence that different defects are responsible for SPEG and for diffusion.

Implications of the nonhydrostatic stress effect. The SPEG rate in Si is enhanced by uniaxial tension and reduced by uniaxial compression, in contrast to the enhancement by hydrostatic pressure. Through an extension of transition state theory to nonhydrostatic stress states, the results were interpreted in terms of a "short and fat" transition state: locally, the system undergoes a contraction in the direction normal to the interface and an in-plane expansion to reach the saddle-point configuration. The uniaxial and hydrostatic results were combined to determine the activation strain tensor for Si SPEG; its symmetry is tetragonal rather than isotropic. The shape of the measured activation strain tensor is inconsistent with the formation or motion of any point defect in the bulk of either phase. With these mechanisms, the measured activation strain tensor is the sum

of a strain of defect formation and a strain of defect migration, and the measurement yields an average over many defects during the growth of many monolayers of material. The average strain of point defect formation must be isotropic in amorphous phases and in cubic crystals. The strain of migration, however, need not be isotropic. Consider, for example, tension in the plane of the (001) interface opening up more space for atoms to migrate toward the interface, enhancing their mobility in the [001] direction only, as shown in Fig. 3(a). This would be consistent with our observation of in-plane tension-enhanced growth rates. However, in this case hydrostatic pressure will correspondingly reduce that space and consequently reduce the mobility, as shown in Fig. 3(b), which is contrary to our observation of pressure-enhanced growth rates. Furthermore, creep measurements²⁷⁻²⁸ indicate that the stresses should be fully relaxed in the amorphous phase in our experiments. For bulk amorphous point-defect mechanisms under these circumstances, wafer-bending cannot affect the growth rate at all. The c/a interface, with its reduced symmetry relative to that of the crystal, is the only place where the opposite-signed effects of uniaxial and hydrostatic compression can be reconciled. Hence the rate-limiting step for SPEG must occur at the interface.

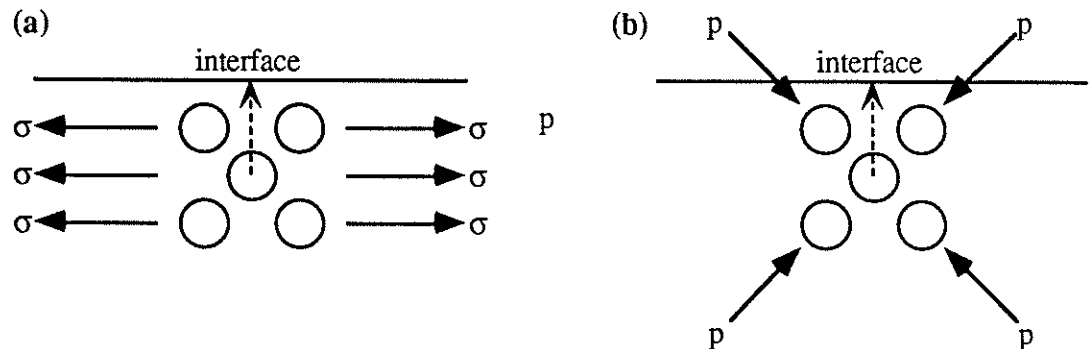


FIG. 3. (a) In-plane tension can enhance mobility of bulk point defects toward interface; (b) in this case, however, hydrostatic pressure reduces mobility toward interface, contrary to experiment.

Implications of the orientation dependence. The SPEG rate in Si varies by about a factor of 20 as the orientation is varied, being fastest if the interface is oriented near (100) and slowest if it is oriented near (111). Since diffusion in both the cubic crystal and the amorphous phase is necessarily isotropic, this observation alone rules out pure bulk-diffusion-controlled mechanisms (see equation (3)) and implies at least some significant role for interface kinetics in determining the overall SPEG rate. Some models combine bulk defect diffusion with interface attachment kinetics in limiting the overall rate. Any such combination would be inconsistent with the observed constant activation energy over 10 decades in growth rate, and with the observed constant activation volume over a more limited velocity range, unless the bulk diffusion process and the interfacial reaction process happen to have identical activation energies and volumes.

Kinetic analysis of the dangling bond mechanism. Taken together, the results make the Spaepen-Turnbull mechanism¹², in which dangling bonds are generated at the interface and migrate along the interface reconstructing the random network into the crystalline network, one of the two proposed mechanisms that remain tenable. The other is the charged kink-site model of Williams and Elliman¹³, which will be discussed separately. The dangling-bond model in addition seems highly plausible because it predicted a negative activation volume²⁹: the mobility of dangling bonds

is enhanced by pressure through a transition state with a lower local volume, in which the dangling bond reaches across a ring to attack a fully-coordinated Si or Ge. A kinetic analysis of the model was undertaken by Lu *et al.* as a further test of its plausibility. It assumes thermal generation of dangling bonds at ledges along the interface, independent migration of the dangling bonds along the ledges to reconstruct the network from the amorphous to the crystalline structure, and unimolecular annihilation kinetics at dangling bond "traps", the exact nature of which remains open. The model yields

$$v = 2 \sin(\theta) v_s n_r \exp\left(\frac{\Delta S_f + \Delta S_m}{k}\right) \exp\left(-\frac{\Delta H_f + \Delta H_m}{kT}\right), \quad (5)$$

where ΔS_f and ΔH_f are the standard entropy and enthalpy of formation of a pair of dangling bonds, ΔS_m and ΔH_m are the entropy and enthalpy of motion of a dangling bond at the interface, v_s is the speed of sound, θ is the misorientation from {111}, and n_r is the net number of hops made by a dangling bond before it is annihilated. It accounts semi-quantitatively for the measured prefactor, orientation dependence, activation energy and activation volume of v , and the pressure of a "free-energy catastrophe" beyond which the exponential pressure-enhancement of SPEG cannot continue uninterrupted due to a vanishing barrier to dangling bond migration. The enhancement of v by doping can be accounted for by an increased number of charged dangling bonds, with no change in the number of neutrals, at the interface. The pressure of the free-energy catastrophe was predicted to be in the range 5-12 GPa in Si, and 3-6 GPa in Ge. The observation by Shimomura *et al.*³⁰ at room temperature of a structural transition to a metallic phase at 10 GPa in a-Si and at 6 GPa in a-Ge may be a manifestation of the free-energy catastrophe.

The structural aspect of the Williams-Elliman kink model^{13,31} seems to be a special case of the Spaepen-Turnbull dangling bond model, where the dangling bonds reside at kink sites on ledges separating {111} terraces. All sites on a flat (100) c/a interface are already at such ledges, and (based on a criterion of counting the number of bonds a crystallizing atom would immediately make to neighbors in the crystal) kinks on those ledges do not offer preferred sites for attachment of atoms in the crystalline configuration. Furthermore, since atoms in the crystal and the amorphous phase are expected to be for the most part fully coordinated, the only way that a kink can move is by a bond rearrangement process. This would almost certainly be brought about by the breaking of bonds across the interface to create pairs of dangling bonds, the rearrangement of the network using the dangling bonds, and the recombining of the dangling bonds. One might then interpret the kink site model as the special case of the dangling bond model in which n_r , the number of jumps of a dangling bond before annihilation, takes its minimum possible value.

Doping Dependence. Although the Spaepen-Turnbull interfacial dangling bond model does not address the dopant-induced enhancement of the SPEG rate directly, we note that the population or mobility of charged dangling bonds at the interface could respond to the doping level through a variety of schemes discussed in the literature^{6,13,15,17,20}. The details are still a matter of debate. Lu *et al.* summarize, critique, and generalize a number of models, keeping in mind that the assumptions about the structural identities of the defects and those about the doping-induced enhancements of their populations or mobilities are in many cases independent of each other and need not stand or fall together. The models that appear most successful at this time are those invoking an enhancement in the concentration of charged defects with doping. Currently at issue is whether the response of the concentration of charged defects to the doping level can be computed using the electronic structure of the crystal or of the amorphous phase.

There have been numerous experimental studies of the doping-induced enhancement of the

SPEG rate and the related compensation effect, in which equal amounts of donors and acceptors offset each other resulting in a growth rate characteristic of undoped material. The Walser group^{32,33} has performed the most thorough experimental investigation to date. They found experimentally that for B, P, or As (but not Al) in (100) Si the concentration-dependence of the SPEG rate can under some circumstances be expressed as

$$v(N_D, T) = v_i(T) \left(1 + \frac{N_D}{N_{\text{norm}}(T)}\right), \quad (6)$$

where N_D is the dopant concentration at the interface, $v_i(T)$ is the growth velocity in intrinsic Si, and the proportionality factor $N_{\text{norm}}(T)$ has Arrhenius form,

$$N_{\text{norm}}(T) = N_0 \exp - \left(\frac{Q_N}{kT}\right), \quad (7)$$

with a temperature-dependence, Q_N , of only ~ 0.3 eV and a prefactor, N_0 , of approximately $3 \times 10^{21}/\text{cm}^3$. This relation holds for concentrations spanning about a decade centered approximately around $10^{19}/\text{cm}^3$ when the interface is between the peak of the implant profile and the free surface. That in many cases, different results are obtained when the interface is deeper than the implant peak is troubling. It seems doubtful, however, that these data will be improved upon unless samples are prepared for which the depth-distribution of dopants is constant, rather than the near-Gaussian profiles produced by ion implantation.

At low concentrations the data can be accounted for by either the Fractional Ionization model of Walser and Jeon²⁰, or by the Fermi-Level-Shifting model of Williams and Elliman¹³ generalized by Lu *et al.*¹⁹ The electric field models of Licoppe and Nissim¹⁷ and of Mosley and co-workers¹⁵ predict too small an effect when developed properly; this seems to be an insurmountable difficulty of these models. The viable models for the doping effect seem to be the generalized Fermi-level-shifting model and the fractional-ionization model, in that they both can be made to reproduce eqs. (6) and (7). They are based on entirely different assumptions. The FLS model assumes that the population of charged defects responds to the density of states of the crystal; the FI model assumes that it responds to the density of states of the amorphous phase. The main problem of the FLS model is that it depends on a defect level that is difficult to find in an independent experiment; also its quantitative predictions are sensitive to the temperature-dependence of the defect level. The main problem with the FI model is that it cannot predict both eq. (6) and the compensation effect. Additional challenges for both models are a prediction of the behavior at concentrations high enough that eq. (6) breaks down, and an explanation of the growth behavior asymmetry between the shallow and deep sides of implanted profiles.

Ion-beam enhanced SPEG. Ion irradiation may affect v by altering the populations of interfacial dangling bonds or may involve bulk point defects of any type impinging on the interface and converting to dangling bonds. Jackson³⁴ models the process as the immediate production of a certain amount of amorphous material during an ion cascade, followed by enhanced crystallization mediated by a radiation-enhanced concentration of point defects which undergo bimolecular annihilation. The structural nature of the defects in Jackson's model need not be specified, but he speculates that they may be dangling bonds. Bimolecular annihilation kinetics are essential to the model in order to obtain any temperature dependence of the ion flux at which the interface does not move. Within the context of the Jackson model, this temperature dependence (1.2 eV) is that of defect motion, ΔE_m . A consequence of bimolecular annihilation kinetics is that the measured

activation energy for thermal SPEG (2.7 eV) is the sum of the energy of motion of a single defect and half the energy of formation of a pair of defects. This is the reason for Jackson's observation that if the same defects responsible for thermal SPEG are to be responsible for beam-enhanced SPEG, his model requires a formation energy of 3 eV per defect pair in Si. This value seems far too large for a bond energy in Si. A transition is possible, from bimolecular annihilation kinetics occurring at the high dangling bond densities resulting from collision cascades, to unimolecular annihilation kinetics in thermal SPEG when the ion beam is reduced and dangling bonds are far less numerous. In this case, Jackson's identification of 1.2 eV as the migration energy still stands, and the bond energy would have to be 1.5 eV. This value seems slightly low, although in a covalent network with significant bond-angle distortion, structural relaxation around the newly-formed dangling bonds might lower their formation energy to this value^{19,35}.

It is also possible that *different* defects are responsible for thermal SPEG and beam-enhanced SPEG. Experiments comparing the effects of channeled and random ion beams on the SPEG rate³⁶ have been interpreted to imply a contribution from bulk crystal point defects. However, any potential role of bulk point defects from either phase in beam-enhanced SPEG has been limited by other similar experiments^{8,37} and by the lack of an observed amorphous-layer thickness dependence to the rate during ion bombardment⁸. Our work on thermal SPEG implies that ion beam-enhanced SPEG may involve, for example, bulk point defects of any type impinging on the interface and converting to interfacial dangling bonds. But when the ion beam is turned off, the rate of interface motion cannot be limited by the arrival rate of these suddenly-less-numerous defects. Ion-beam induced bulk point-defect mechanisms, operating *in parallel* with thermal generation of dangling bonds at the interface, are also possible. For any of these scenarios to be given much credence, the transition from beam-enhanced SPEG to thermal SPEG as the ion flux is lowered must be addressed quantitatively.

Conclusions

The conclusions for thermal SPEG are summarized in Table 1. Bulk crystal point-defect mechanisms are untenable because of the magnitude and sign of the activation volume for SPEG and because of the low bulk crystal diffusivity. Bulk amorphous point-defect mechanisms are untenable because the relaxation behavior for SPEG is different than that for diffusion. All bulk point defect mechanisms are untenable because uniaxial compression reduces v while hydrostatic pressure enhances v . All bulk point defect mechanisms are also inconsistent with the combination of the orientation-dependence and the constant activation energy and volume of v . The experiments are consistent with any mechanism involving defect formation or motion at the interface. A kinetic analysis of the Spaepen-Turnbull dangling bond mechanism, postulating thermal generation of dangling bonds at the interface and unimolecular annihilation kinetics at "traps", shows it to be a highly plausible model for the growth process. The nature of the "traps" is at present unclear. The Williams-Elliman kink model seems to be a special case of the dangling bond model, where the dangling bonds reside at kink sites and the number of jumps of a dangling bond before annihilation takes its minimum possible value. Within the context of the kinetic analysis of Lu *et al.*, the number of atoms crystallized per dangling bond formed is between 5 and 200 in Si, and 30 and 1200 in Ge, which makes the limiting case of the kink model seem somewhat low. For both Si and Ge the model gives plausible semi-quantitative interpretations to the prefactor, orientation dependence, activation energy, and activation volume of SPEG, and to the maximum pressure attainable before a "free energy catastrophe" eliminates the barrier to motion of dangling bonds.

The doping-dependence might be explained by the density of states of the amorphous phase through the fractional-ionization model of Walser and Jeon, or by the density of states of the crystal through a generalized Williams-Elliman Fermi-level shifting model. As we have shown that the relevant defects reside at the *c/a* interface, it might now be appropriate to call for a third type of doping model based on the density of states *in the interface*.

These conclusions are valid for thermal SPEG only. Ion irradiation may alter the populations of dangling bonds in a manner described by the Jackson model, with a transition from bimolecular to unimolecular annihilation kinetics as the ion beam is turned off. The work of Lu *et al.* on thermal SPEG implies that ion beam-enhanced SPEG may involve, for example, bulk point defects of any type impinging on the interface and converting to interfacial dangling bonds. But when the ion beam is turned off, the rate of interface motion cannot be limited by the arrival rate of these suddenly-less-numerous defects. It may also involve alternative point-defect mechanisms operating *in parallel* with thermal generation of dangling bonds at the interface.

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